

# PULMONARY EMBOLISM: DIAGNOSIS BY CHEST LEAD ELECTROCARDIOGRAPHY

BY

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Limb lead electrocardiograms in cases of pulmonary embolism show characteristic changes, which have been carefully studied by Barnes (1937). The essential features are a tendency towards right axis deviation, with a constant S wave in lead I, and in lead III a moderate Q wave and sharp inversion of T. The appearances in lead III simulate posterior myocardial infarction, although Q is inconstant, and the R-T segment is rarely elevated and never markedly so. Fig. 1 shows three serial cardiograms from a case of massive pulmonary embolism proved at autopsy. During life this patient, who was originally put

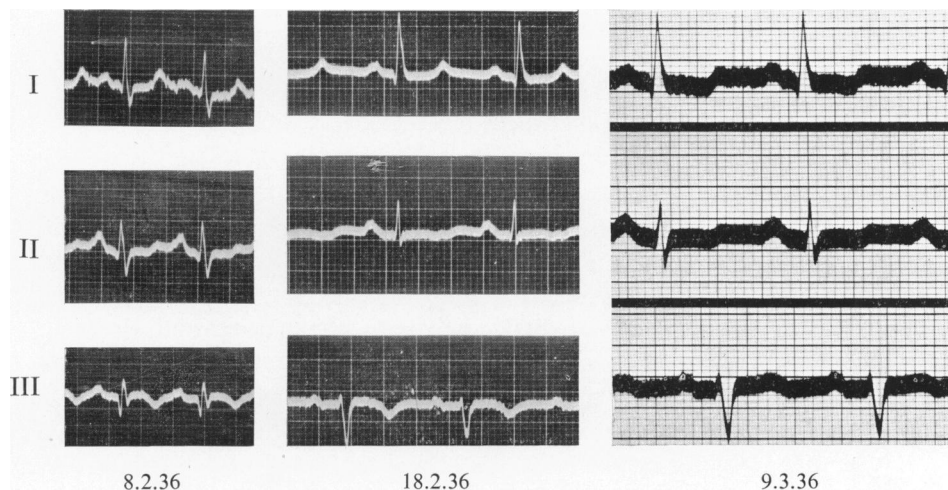


FIG. 1.—Serial limb lead electrocardiograms in a case of pulmonary embolism proved at autopsy. (The case was misdiagnosed as one of posterior infarction during life.) Case 1.

to bed for a rest on account of hypertensive heart disease, was wrongly diagnosed as posterior myocardial infarction. The differential diagnosis between these two conditions may usually be made by giving consideration to all the points tabulated by Barnes, but there are times when the distinction is difficult, if not

impossible. Further, on the clinical side, although as a rule the diagnosis of one or the other is not in doubt, there are occasions when there appear to be no distinguishing features. Substernal pain, a feeling of having been struck in the chest, tightness in the chest, breathlessness, faintness or loss of consciousness, profuse sweating, and prostration are symptoms common to both; while a small rapid pulse, a fall of blood pressure, gallop rhythm, and a cold, clammy, grey skin are signs common to both. Radiation of pain to the neck or arms, a change of cardiac rhythm, or true pericardial friction favour myocardial infarction; whereas an abrupt or early rise of venous blood pressure points to pulmonary embolism. Finally, in a series of 289 cases of pulmonary embolism found at autopsy to be the cause of post-operative death, a series in which as high a proportion as 82 per cent were correctly diagnosed in life, coronary occlusion headed the list of incorrect diagnoses (Nygaard, 1938). In another autopsy study of post-operative pulmonary emboli about two thirds of 229 cases were not diagnosed clinically (Pretin, 1936). Of pertinent interest is the series of 200 cases of coronary thrombosis reported by Eppinger and Kennedy (1938), for pulmonary embolism was found to be the cause of death in 6 per cent, and was present in 24 per cent. Belt (1939) especially has drawn attention to the fact that pulmonary emboli are as common amongst the patients in the medical wards as amongst those in surgical wards.

Enough has been said to make it clear that a more certain method of diagnosing pulmonary embolism is required, and that it should leave no room for confusion in distinguishing pulmonary embolism from posterior myocardial infarction. It is the object of this paper to present such a method.

#### CHEST LEAD ELECTROCARDIOGRAMS IN 10 CASES

Chest lead electrocardiograms have been used widely as an aid in the diagnosis of myocardial infarction. As we are concerned here only with the difficulty of distinguishing pulmonary embolism from posterior infarction, it is only necessary to refer to the chest lead appearances of such infarcts. As is now well known there may be no changes recorded, or there may be depression of the RS-T segment with an upright T wave, or there may be very tall T waves, but there is never inversion of the T wave in any chest lead (Wood and Selzer, 1939). Examples of the two latter changes are shown in Figs. 2 and 3 (p. 24).

During the last few years at Hammersmith Hospital chest lead cardiograms have been taken on all suspected cases of pulmonary embolism and have been found very helpful. Ten cases are presented in Table I, compiled in order to show upon what evidence the diagnosis was made. Only those cases with considerable obstruction of the pulmonary circulation have been included, for it was found that small emboli insufficient to throw any stress on the right ventricle produced no changes in the electrocardiogram and in no way imitated myocardial infarction. Further, all these cases necessarily lived long enough for serial cardiograms to be taken, with one exception that is only included

TABLE I.—DETAILS OF THE TEN CASES

Case No.	Sex and Age	Diagnosis and Cause of Embolism †	Clinical Features: all showed Dyspnoea and Pallor													Proof of Em- bolism	
			Recurrent Attacks	Pain } Tightness } Substernal	Syncope	Blood Pressure	Pulse Rate	Sweats	Cyanosis	Engorged Cervical Veins	Hepatic Enlargement	Right Ventricular Gallop	Pulmonary Consolidation (C), Haemoptysis (H), or X-Ray Opacity (X)	Pleural Pain	Pleural Friction		Haemorrhagic Pleural Effusion
1	F., 63	Hypertensive heart disease	+	+	—	145	95	+	++	+	+	+	H	—	—	—	A
2*	M., 63	Appendicectomy, 10th day †	+	+	+	80	90	+	++	+	+	—	—	+	—	—	C
3	F., 38	Pyelitis †	—	+	+	70	144	+	+	+	+	+	H	+	+	+	C
4	M., 49	Perforated duodenal ulcer: post-operative †	—	—	—	90	140	+	+	+	+	+	CH	+	+	—	C
5	M., 35	Gastric ulcer: post-operative	—	—	—	100	130	—	+	+	+	+	CX	—	—	—	C
6*	M., 20	Mild injury to leg †	—	—	+	80	115	+	+	+	+	+	X	—	—	—	C
7*	F., 34	Cervicitis †	—	—	+	140	120	+	++	+	—	—	—	—	—	—	—
8	F., 53	Carcinoma of rectum	—	—	+	80	—	+	++	+	+	+	—	—	—	—	OA
9	F., 43	Partial hysterectomy for fibroids	+	+	+	60	150	+	—	+	—	—	—	—	—	—	OA
10	M., 25	Mitral stenosis	+	—	—	60	95	+	+	+	+	+	H	—	—	—	A

\* Not seen by the author within three days of the onset.

† Deep femoral venous thrombosis, as evidenced by oedema of the limb, was the cause in all these cases except in Case 7, where it was due to a pelvic injection of anæsthetic oil, proctocaine.

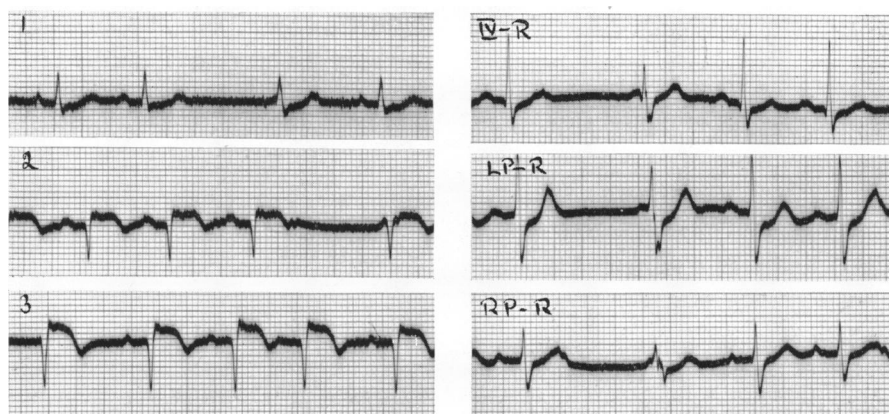


FIG. 2.—Depression of the RS-T segment is seen in the left pectoral lead in this case of posterior myocardial infarction.

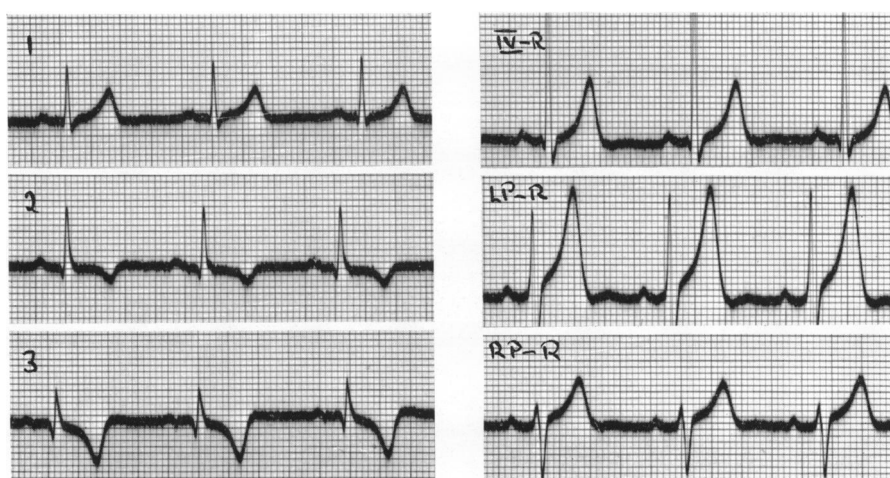


FIG. 3.—Posterior myocardial infarction with very tall T waves in the left pectoral lead.

to show that the changes take time to develop. I have encountered no reasonably proved case of pulmonary embolism with evidence of right ventricular stress that failed to show the changes about to be described, provided life was not extinguished too quickly.

Multiple chest lead cardiograms were taken from the apex beat (lead IV), from the fourth right intercostal space at the right border of the sternum (right pectoral : RP-R), and from a point midway between these two (left pectoral : LP-R). The illustrations show lead IV on top, the left pectoral lead below it, and the right pectoral lead at the bottom (Wood and Selzer, 1939). The proximal electrode was always paired with the right-arm electrode, so that the apical lead was lead IV R.

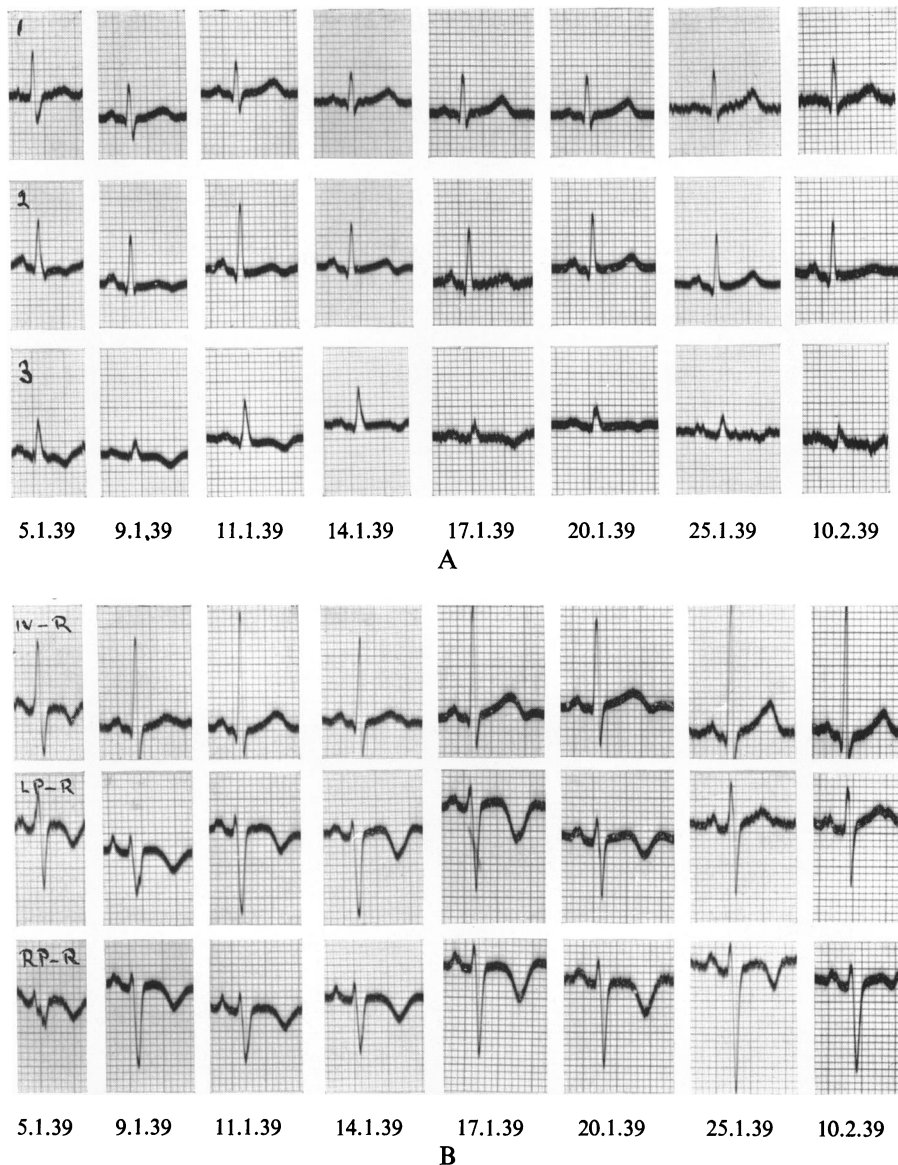


FIG. 4.—Serial electrocardiograms from a case of pulmonary embolism (Case 4).  
 (A) Limb leads, showing changes in T<sub>2</sub> of short duration and in T<sub>3</sub> of longer duration.  
 (B) Chest leads, showing T inversion of long duration in LP-R and RP-R.

Fig. 10 (Wood and Selzer, 1939) and Figs. 4 and 5 show the diagnostic changes discovered in three typical cases (Cases 3, 4, and 5). The essential feature is sharp inversion of the T wave, without appreciable displacement of the RS-T segment, always in the right pectoral lead, usually but for a shorter duration in the left pectoral lead, and sometimes and for the shortest duration in lead IV. Less essential is a tendency for the QRS deflection to be mainly upwards in the right pectoral lead. The T wave commonly remains inverted

for several weeks in the right pectoral lead, for a week or two in the left pectoral lead, and for a day or two, if at all, in lead IV. These facts may be verified by examination of the dates of the serial cardiograms in the examples shown. It is, however, easier to follow the stages of recovery than the stages of develop-

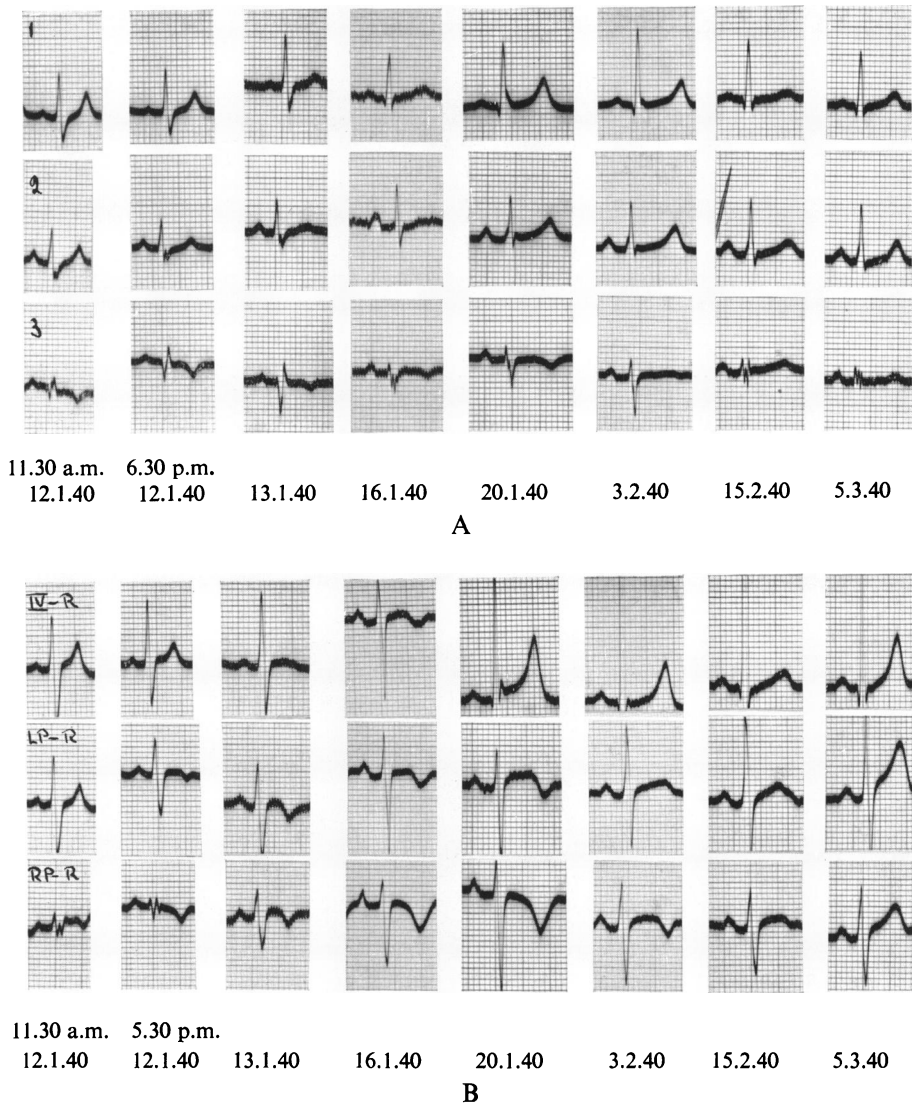


FIG. 5.—Serial electrocardiograms from a case of pulmonary embolism (Case 5).

(A) Limb leads, showing  $T_s$  inversion.

(B) Chest leads, showing the maximum change about the fourth to eighth day.

ment. Case 8 died one hour after the event, and the cardiogram that was taken within a half hour shows no change. In Case 5 (Fig. 5) the initial cardiogram was recorded within a few hours after the onset, and it will be seen that changes are minimal; six hours later they are still developing, and on the following day

they are established. Only regressive changes were observed in all the other cases, although in six of the eight the first cardiogram was taken within six to twenty-four hours after the onset. It appears, therefore, that maximal changes occur in a few hours but are not immediate.

#### DISCUSSION

It has been argued that the limb lead changes indicate myocardial ischæmia because they imitate those of posterior myocardial infarction (Parsons-Smith, 1940). This ischæmia is thought to be due to shock, to anoxæmia, or to reflex coronary vaso-constriction. Further, Scherf and Boyd (1939) point out that the right ventricle would be especially embarrassed by myocardial ischæmia because of the burden thrown upon it by the obstruction in the pulmonary circulation. These views are open to criticism. First, although the limb lead cardiogram may imitate the features of posterior myocardial infarction, the chest lead appearances are entirely different; this argument must therefore lapse. Second, it has not been shown that shock alone is capable of producing cardiographic changes of the kind under discussion. While it is not denied that shock may influence the symptoms of pulmonary embolism, there is no reason to believe that it is any more responsible for the cardiographic changes than it is in cases of myocardial infarction. Third, reflex coronary vaso-constriction is said to be independent of the size of the pulmonary embolus (Scherf and Schönbrunner, 1937): these authors described two cases with marked cardiographic changes following small pulmonary emboli, and witnessed a typical electrocardiograph pattern as a result of small experimental emboli in three out of ten dogs.

Yet, in the present series, the cardiographic changes appeared to depend very much upon the size of the embolus, and ran parallel to the development of acute cor pulmonale. Reference to the table will show that engorgement of the cervical veins was noted in seven out of the ten cases, and in each of these I made the observation myself within twenty-four hours of the onset. In the other three I failed to see the patient before at least three days had elapsed, and it is well known that the sign is apt to be overlooked unless special attention is paid to it. I have carefully examined many cases of pulmonary embolism within twenty-four hours of the event, and have not seen the cardiographic changes described above in the absence of evidence of acute cor pulmonale. It follows that, in the majority of cases, coronary vaso-constriction cannot be held responsible for these cardiograms unless it be associated with acute cor pulmonale. But if acute right ventricular stress from any cause can be shown to produce a similar cardiographic pattern, then it would seem that the theory of coronary vaso-constriction is not required to explain them. This appears to be the case. Selzer and I (1939) have recorded similar features, but in a persistent form, in pulmonary stenosis, mitral stenosis, and chronic cor pulmonale; transient changes, indistinguishable from those produced by pulmonary embolism, are found in rheumatic carditis (Wood, 1939), diphtheria

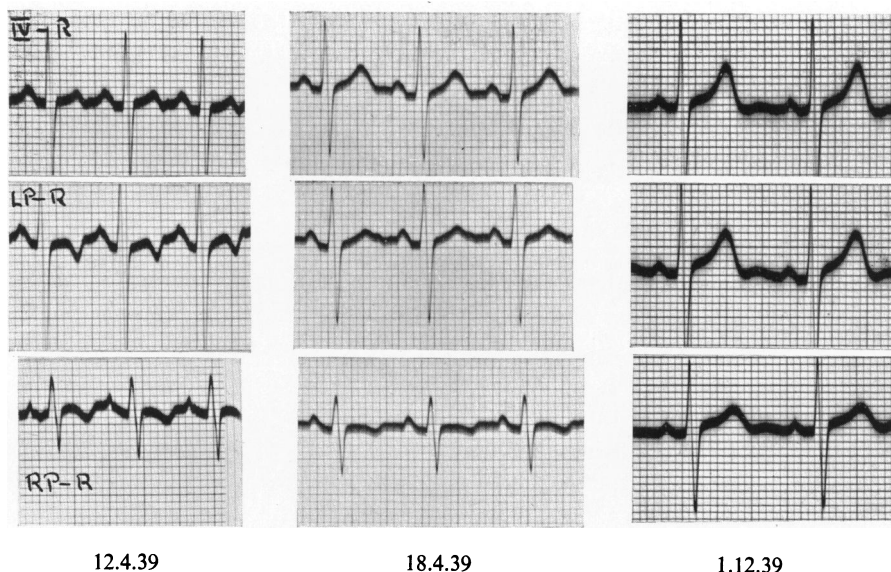


FIG. 6.—Limb and chest lead electrocardiograms from a case of pneumonia. The serial chest lead records are indistinguishable from those of pulmonary embolism. The standard leads taken on the same dates showed some inversion of T in lead III which gradually diminished and disappeared.

(Pincus, 1939), and pneumonia (see Fig. 6), conditions known to be associated with isolated right ventricular failure.

It is concluded that the essential factor in the production of these changes is right ventricular stress. It is not unlikely that the explanation when discovered will be the same as that for T wave inversion in leads I and IV in cases of left ventricular stress, e.g. in hypertensive heart disease, when T in lead I inversion may be found associated with widely dilated coronary arteries (Harrison and Wood).

The same argument may be used to show that the chest lead electrocardiograms described, although providing a good method for the differential diagnosis between pulmonary embolism and posterior myocardial infarction, are not in themselves diagnostic of pulmonary embolism, but of acute and transient right ventricular stress. They bear the same significance as does a rise in systemic venous blood pressure, but whereas the latter may last only a few hours or days, the cardiographic pattern usually persists for weeks.

#### SUMMARY

1. Acute pulmonary embolism may be difficult to distinguish from posterior myocardial infarction, both clinically and by means of limb lead electrocardiograms.
2. Multiple chest lead cardiograms afford a good method of differential diagnosis.
3. In posterior myocardial infarction, as is well known, there may be no cardiographic change, or the RS-T segment may be depressed, or the T waves may be very tall.



4. In pulmonary embolism sufficient to cause right ventricular stress there is sharp inversion of the T wave, maximal and for the longest duration in the right pectoral lead; usually, but for a shorter duration, in the left pectoral lead; and rarely, and for the shortest duration, in lead IV.

5. Similar changes may be found in all conditions giving rise to right ventricular stress.

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